WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences

Search Results -

Terms	Documents
l1 near20 l2	26

Database: US Patents Full Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: `l1 near20 l2` Clear

Search History

Today's Date: 6/7/2000

DB Name	Query	Hit Count	Set Name
USPT,JPAB,EPAB,DWPI	l1 near20 l2	26	L4
USPT,JPAB,EPAB,DWPI	l1 same l2	148	L3
USPT,JPAB,EPAB,DWPI	etanercept or infliximab or (tnf$2 or tumor adj necrosis adj factor$2 or anti-tnf$3) near4 (antagonist$1 or inhibit$3 or receptor$1 or antibod$3) or cdp571 or d2e7	3187	L2
USPT,JPAB,EPAB,DWPI	(neurological or neurodegenerat$3 or spinal adj cord or brain) near3 (condition$1 or disease$1 or damage or trauma$1 or injur$3 or disorder$1 or tumor$1) or alzheimer$2 or huntington$2 or creutzfeld$ or parkinson$2 or myasthenia or guillain$6 or bell$2 adj palsy or neuropath$3	44664	L1

```
FILE 'HOME' ENTERED AT 14:07:15 ON 07 JUN 2000

=> file reg

COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.30       0.30

FILE 'REGISTRY' ENTERED AT 14:08:28 ON 07 JUN 2000
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2000 American Chemical Society (ACS)

STRUCTURE FILE UPDATES:     6 JUN 2000  HIGHEST RN 268568-90-7
DICTIONARY FILE UPDATES:    6 JUN 2000  HIGHEST RN 268568-90-7

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 11, 2000

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Structure search limits have been increased.  See HELP SLIMIT
for details.

=> e etanercept/cn

E1            1      ETANAUTINE/CN
E2            1      ETANDAN/CN
E3            1 --> ETANERCEPT/CN
E4            1      ETANIDAZOLE/CN
E5            1      ETANOR/CN
E6            1      ETANTEROL/CN
E7            1      ETAP/CN
E8            1      ETAPAK/CN
E9            1      ETAPERAZIN/CN
E10           1      ETAPERAZINE/CN
E11           1      ETAPHEN/CN
E12           1      ETAPHOS/CN

=> s e3

L1            1 ETANERCEPT/CN

=> d


L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2000 ACS
RN   185243-69-0  REGISTRY
CN   1-235-Tumor necrosis factor receptor (human) fusion protein with
     236-467-immunoglobulin G1 (human .gamma.1-chain Fc fragment) (9CI)  (CA
     INDEX NAME)
OTHER NAMES:
CN   Embrel
CN   Enbrel
CN   Etanercept
CN   rhu TNFR:Fc
```

```
FS   PROTEIN SEQUENCE
DR   200013-86-1
MF   Unspecified
CI   MAN
SR   US Adopted Names Council
LC   STN Files:   ADISINSIGHT, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS,
       DRUGNL, DRUGPAT, DRUGUPDATES, EMBASE, IPA, MEDLINE, MRCK*, PHAR,
       TOXLINE, TOXLIT, USPATFULL
         (*File contains numerically searchable property data)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
              20 REFERENCES IN FILE CA (1967 TO DATE)
              21 REFERENCES IN FILE CAPLUS (1967 TO DATE)


=> e infliximab/cn

E1            1     INFLEXUSIN/CN
E2            1     INFLEXUSIN B/CN
E3            1 --> INFLIXIMAB/CN
E4            1     INFLUINA/CN
E5            1     INFLUMIN/CN
E6            1     INFO 1/CN
E7            1     INFO 2/CN
E8            1     INFO 5/CN
E9            1     INFO 531/CN
E10           1     INFOLITE ER 51/CN
E11           1     INFONUTROL/CN
E12           1     INFORM 6350M/CN

=> s e3

L2            1 INFLIXIMAB/CN

=> d


L2   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2000 ACS
RN   170277-31-3  REGISTRY
CN   Immunoglobulin G, anti-(human tumor necrosis factor) (human-mouse
     monoclonal cA2 heavy chain), disulfide with human-mouse monoclonal cA2
     light chain, dimer (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Avakine
CN   Infliximab
CN   Remicade
MF   Unspecified
CI   MAN
SR   US Adopted Names Council
LC   STN Files:   ADISINSIGHT, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS,
CIN,
       DRUGNL, DRUGPAT, DRUGUPDATES, EMBASE, IMSDIRECTORY, IPA, PROMT,
TOXLINE,
       TOXLIT, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
              25 REFERENCES IN FILE CA (1967 TO DATE)
              27 REFERENCES IN FILE CAPLUS (1967 TO DATE)


=> e cdp571/cn

E1            1     CDP-STAR/CN
E2            1     CDP-TYVELOSE EPIMERASE/CN
```

```
E3            0 --> CDP571/CN
E4            1     CDPA/CN
E5            1     CDPASE/CN
E6            1     CDPC 3510/CN
E7            1     CDPDIGLYCERIDE-INOSITOL PHOSPHATIDYLTRANSFERASE/CN
E8            1     CDPK KINASE/CN
E9            1     CDPK-RELATED PROTEIN KINASE/CN
E10           1     CDPK-RELATED PROTEIN KINASE (CORN CLONE ZMCRK1 C-TERMINAL
FR
                    AGMENT)/CN
E11           1     CDPK-RELATED PROTEIN KINASE (CORN CLONE ZMCRK3)/CN
E12           1     CDPPOET/CN

=> e cdp-571/cn

E1            1     CDP-4-KETO-3,6-DIDEOXY-D-GLUCOSE 4-REDUCTASE/CN
E2            1     CDP-4-KETO-6-DEOXY-D-GLUCOSE-3-DEHYDRASE/CN
E3            0 --> CDP-571/CN
E4            1     CDP-6-DEOXY-.DELTA.3,4-GLUCOSEEN REDUCTASE/CN
E5            1     CDP-6-DEOXY-D-GLYCERO-L-THREO-4-HEXULOSE-3-DEHYDRATASE/CN
E6            1     CDP-6-DEOXY-D-XYLO-4-HEXULOSE 3-DEHYDRASE/CN
E7            1     CDP-6-DEOXY-DELTA-3,4-GL UCOSEEN REDUCTASE (NEISSERIA
MENING
                    ITIDIS STRAIN MD58 GENE NMB1359)/CN
E8            1     CDP-6-DEOXY-L-THREO-D-GLYCERO-4-HEXULOSE-3-DEHYDRASE/CN
E9            1     CDP-6-DEOXY-L-THREO-D-GLYCERO-4-HEXULOSE-3-DEHYDRASE
REDUCTA
                    SE/CN
E10           1     CDP-6-DEOXY-L-THREO-D-GLYCERO-4-HEXULOSE-3-DEHYDRATASE/CN
E11           1     CDP-ABEQUOSE/CN
E12           1     CDP-ABEQUOSE SYNTHASE/CN

=> e cdp 571/cn

E1            1     CDP 1012/CN
E2            1     CDP 25/CN
E3            0 --> CDP 571/CN
E4            1     CDP 713/CN
E5            1     CDP 840/CN
E6            1     CDP 845/CN
E7            1     CDP 9/CN
E8            1     CDP ABEQUOSE EPIMERASE/CN
E9            1     CDP DISODIUM SALT/CN
E10           1     CDP GLYCEROL PYROPHOSPHATASE/CN
E11           1     CDP II/CN
E12           1     CDP KINASE/CN

=> e d2e7/cn

E1            1     D280/CN
E2            1     D289/CN
E3            0 --> D2E7/CN
E4            1     D2EHPA SODIUM SALT/CN
E5            1     D2F/CN
E6            1     D2S/CN
E7            1     D2SV/CN
E8            4     D3/CN
E9            1     D3 STEEL/CN
E10           1     D3-METHYLTRICHLOROSILANE/CN
E11           1     D3-VIGANTOL/CN
E12           1     D30/CN
```

```
 * * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'CA, BIOSIS, MEDLINE, DRUGU, EMBASE'
AT 14:14:19 ON 07 JUN 2000
FILE 'CA' ENTERED AT 14:14:19 ON 07 JUN 2000
COPYRIGHT (C) 2000 AMERICAN CHEMICAL SOCIETY (ACS)
FILE 'BIOSIS' ENTERED AT 14:14:19 ON 07 JUN 2000
COPYRIGHT (C) 2000 BIOSIS(R)
FILE 'MEDLINE' ENTERED AT 14:14:19 ON 07 JUN 2000
FILE 'DRUGU' ENTERED AT 14:14:19 ON 07 JUN 2000
COPYRIGHT (C) 2000 DERWENT INFORMATION LTD
FILE 'EMBASE' ENTERED AT 14:14:19 ON 07 JUN 2000
COPYRIGHT (C) 2000 Elsevier Science B.V.  All rights reserved.

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     3.35      14.89

=> s l1 or l2 or etanercept or infliximab or (tnf? or tumor necrosis factor?
or anti-tnf?)(4a)(receptor? or antagonist? or antibod? or inhibit?)
   2 FILES SEARCHED...
   4 FILES SEARCHED...
L3       46298 L1 OR L2 OR ETANERCEPT OR INFLIXIMAB OR (TNF? OR TUMOR
NECROSIS
               FACTOR? OR ANTI-TNF?)(4A)(RECEPTOR? OR ANTAGONIST? OR ANTIBOD?
               OR INHIBIT?)

=> s (neurolog? or neurodegenerat? or neuron? or neuromusc?)(3a)\
(condition? or disorder? or disease? or ) or alzheim? or parkinson? or
huntington? or creutzfeld-jakob or myasthen? grav? or guillain-barre or bell?
palsy or
MISSING OPERATOR '\  (CONDITION?'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (neurolog? or neurodegenerat? or neuron? or neuromusc? or brain or
spinal cord or cns or nerv?)(3a)(condition? or disorder? or disease? or
trauma? or injur? or tumor? or lesion?) or alzheim? or parkinson? or
huntington? or creutzfeld-jakob or myasthen? grav? or guillain-barre or bell?
palsy
 PREVIOUS MSG TOO LONG
<------User Break------>

=> s (neurolog? or neurodegenerat? or neuron? or neuromusc? or brain or
spinal cord or cns or nerv?)(3a)(condition? or disorder? or disease? or
trauma? or injur? or tumor? or lesion?) or alzheim? or parkinson? or
huntington? or creutzfeld-jakob or myasthen? grav? or guillain-barre
   2 FILES SEARCHED...
   3 FILES SEARCHED...
L4      849335 (NEUROLOG? OR NEURODEGENERAT? OR NEURON? OR NEUROMUSC? OR
BRAIN
               OR SPINAL CORD OR CNS OR NERV?)(3A)(CONDITION? OR DISORDER? OR
               DISEASE? OR TRAUMA? OR INJUR? OR TUMOR? OR LESION?) OR
ALZHEIM?
               OR PARKINSON? OR HUNTINGTON? OR CREUTZFELD-JAKOB OR MYASTHEN?
               GRAV? OR GUILLAIN-BARRE

=> s bell? palsy or neuropath? or ms or multiple sclero? or panencephalit? or
als or amyotroph?
```

```
L5      490361 BELL? PALSY OR NEUROPATH? OR MS OR MULTIPLE SCLERO? OR
PANENCEPH
               ALIT? OR ALS OR AMYOTROPH?

=> s l3 and (l4 or l5)
L6         2013 L3 AND (L4 OR L5)

=> s l3(l)(l4 or l5)
L7         1519 L3(L)(L4 OR L5)

=> s l1 or l2 or etanercept or infliximab
L8          610 L1 OR L2 OR ETANERCEPT OR INFLIXIMAB

=> s l8 and (l4 or l5)
L9           22 L8 AND (L4 OR L5)

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10          19 DUP REM L9 (3 DUPLICATES REMOVED)

=> d 1-19 bib,ab

L10  ANSWER 1 OF 19  CA  COPYRIGHT 2000 ACS
AN   132:260696  CA
TI   Use of TNF-.alpha. inhibitors for treating nerve root
     injury
IN   Olmarker, Kjell; Rydevik, Bjorn
PA   A+ Science Invest AB, Swed.
SO   PCT Int. Appl., 29 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   WO 2000018409      A1   20000406      WO 1999-SE1671   19990923
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
             CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
             IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD,
             MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK,
             SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRAI SE 1998-3276     19980925
     SE 1998-3710     19981029
AB   Pharmaceutical compns. for the treatment of spinal disorders caused by
the
     liberation of TNF-.alpha. comprise an effective amt. of a TNF-.alpha.
     inhibitor.  Also provided are a method for treatment of such disorders
and
     the use of TNF-.alpha. inhibitors in the prepn. of a pharmaceutical
compn.
     for such treatment.
RE.CNT  8
RE
(2) Olmarker, K; SPINE 1994, V19(16), P1803 MEDLINE
(3) Olmarker, K; SPINE 1998, V23(23), P2538 MEDLINE
(4) Pennica, D; NEURON 1996, V17(1), P63 CA
(7) Sommer, C; NEUROSCIENCE LETTERS 1997, V237(1), P45 CA
(8) Sommer, C; PAIN 1998, V74(1), P83 CA
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10  ANSWER 2 OF 19  CA  COPYRIGHT 2000 ACS
```

AN 132:232740 CA
TI Protein and cDNA sequences of honey bee venom protein PX3.101, and uses thereof in the treatment of various diseases
IN Cui, Xiangmin; Lu, Yuefeng
PA Pan Pacific Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000015774	A1	20000323	WO 1999-US21077	19990913

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1998-PV100172 19980914
AB The invention provides protein and cDNA sequences of a novel protein, PX3.101, which can be isolated from honey bee venom. The invention also provides pharmaceutical compns. based upon PX3.101 polypeptide and methods for using same in the treatment of various diseases, including various inflammatory diseases such as rheumatoid arthritis. The invention further relates to the treatment of diseases assocd. with chemokine (esp. IL-8) imbalances, wherein PX3.101 inhibits the binding of a chemokine with its receptor.
RE.CNT 1
RE
(1) Frei, E; The EMBO Journal 1988, V7(1), P197 CA

L10 ANSWER 3 OF 19 CA COPYRIGHT 2000 ACS
AN 132:73662 CA
TI Tumor necrosis factor antagonists for the treatment of **neurological disorders**
IN Tobinick, Edward L.; Tobinick, Arthur Jerome
PA USA
SO U.S., 7 pp., Cont.-in-part of U. S. Ser. No. 256,388, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6015557	A	20000118	US 1999-275070	19990323

PRAI US 1999-256388 19990224
AB A method is provided for inhibiting the action of TNF for treating neurol. conditions in a human by administering a TNF antagonist for reducing damage to neuronal tissue or for modulating the immune response affecting neuronal tissue of the human. The TNF antagonist administered is selected from the group consisting of **etanercept** and **infliximab** . The TNF antagonist is administered s.c., i.v., intrathecally, or i.m. Methotrexate or Leflunomide may be administered concurrently with the TNF antagonist for demyelinating diseases and certain other neurol. disorders.
RE.CNT 3
RE

(1) Aggarwal; US 5795967 1998
(2) Jacobs; US 5605690 1997
(3) Le; US 5656272 1997 CA

L10 ANSWER 4 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 2000141071 EMBASE
TI [Report from Great Britain].
BERICHT AUS GROSSBRITANNIEN.
AU Woodhouse R.J.
CS R.J. Woodhouse, 4 Swainswick Gardens, Bath BA1 6TL, United Kingdom
SO Pharmazeutische Industrie, (2000) 62/3 (202-206).
ISSN: 0031-711X CODEN: PHINAN
CY Germany
DT Journal; Article
FS 006 Internal Medicine
037 Drug Literature Index
039 Pharmacy
LA German

L10 ANSWER 5 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 2000025166 EMBASE
TI Musculoskeletal and systemic reactions to biological therapeutic agents.
AU Watts R.A.
CS Dr. R.A. Watts, Ipswich Hospital, Heath Road, Suffolk IP4 5PD, United Kingdom. Rwatts@Dial.pipex.com
SO Current Opinion in Rheumatology, (2000) 12/1 (49-52).
ISSN: 1040-8711 CODEN: CORHES
CY United States
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Autoimmune disease, in particular systemic lupus erythematosus (SLE), can be induced by drugs. Over the past couple of years biologic agents have become available for the treatment of inflammatory disease; simultaneously, researchers have realized that these drugs can not only suppress autoimmune disease but may also potentiate it.
Interferon-.alpha.
and interferon-.beta. both may induce autoimmune disease, but this is more
frequent with interferon-.alpha., Therapy to block tumor necrosis factor-.alpha., either with monoclonal anti-bodies or fusion proteins, has
been associated with the development of antinuclear antibodies, but only rarely with clinical development of SLE. None of the three reported cases of SLE occurring after anti-tumor necrosis factor-.alpha. therapy has developed major organ involvement. The continued use of biologic agents will provide interesting insights into the pathogenesis of autoimmune disease.

L10 ANSWER 6 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 2000164376 EMBASE
TI First anniversary editorial.
AU Hagmann W.K.; McMillan R.
CS W.K. Hagmann, Merck Research Laboratories, PO Box 2000, Rahway, NJ 07065-0900, United States
SO Current Opinion in Anti-inflammatory and Immunomodulatory Investigational Drugs, (2000) 2/2 (i-ii).
ISSN: 1464-8474 CODEN: COAIFF
CY United Kingdom
DT Journal; Editorial
FS 037 Drug Literature Index

015 Chest Diseases, Thoracic Surgery and Tuberculosis
008 Neurology and Neurosurgery
031 Arthritis and Rheumatism
LA English

L10 ANSWER 7 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999332664 EMBASE
TI Rheumatoid arthritis: Newest strategies to control the pain.
AU Lipman A.G.
CS Dr. A.G. Lipman, College of Pharmacy, Pain Management Center, Univ. of Utah Health Sciences Center, Salt Lake City, UT, United States
SO Consultant, (1999) 39/4 (1228-1244).
Refs: 14
ISSN: 0010-7069 CODEN: CNSLAY
CY United States
DT Journal; General Review
FS 006 Internal Medicine
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Many patients with rheumatoid arthritis (RA) face a life of chronic pain. Primary care clinicians have the opportunity to intervene early and aggressively with a wide range of pharmacologic and physical modalities to control pain and prevent its deleterious effects- and thus improve quality of life. Options include simple analgesics, NSAIDs, disease-modifying antirheumatic drugs (DMARDs), release of trigger points for myofascial pain syndromes, adjunctive medications for **neuropathic** pain syndromes, and opioids for carefully selected patients. Physical therapy and rehabilitation remain cornerstones in the treatment of RA. The new cyclooxygenase-2 inhibitors and newer DMARDs, such as leflunomide and **etanercept**, are less likely than older agents to produce serious gastrointestinal and other adverse effects.

L10 ANSWER 8 OF 19 DRUGU COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 2000-12565 DRUGU T M S
TI Recent additions to the growing biotechnology armamentarium: a critical assessment.
AU Schrand L M
CS Univ.Iowa
LO Iowa City, Iowa, USA
SO Formulary (34, No. 11, 920-42, 1999) 1 Fig. 8 Tab. 55 Ref.
CODEN: FORMF ISSN: 1082-801X
AV Department of Pharmaceutical Care, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, Iowa City, IA 52242, U.S.A.
LA English
DT Journal
FA AB; LA; CT
FS Literature
AB The biotechnology agents **infliximab**, interferon-alpha-con-1, basiliximab, daclizumab and trastuzumab are reviewed, with respect to their mode of action, clinical trial findings, safety, and place in therapy. Comparisons are made with standard antiinflammatory, virucidal, immunosuppressive or cytostatic therapies, including prednisone, azathioprine, mercaptopurine, ciclosporin, methotrexate, IFN-alpha-2a, IFN-alpha-2b, ribavirin, muromonab-CD3, horse antithymocyte globulin, rabbit antithymocyte globulin ciclosporin, mycophenolate mofetil, paclitaxel, doxorubicin, epirubicin, cyclophosphamide and cisplatin.

L10 ANSWER 9 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999315270 EMBASE
TI Novel therapeutic strategies.

AU Worker C.
CS C. Worker, Current Drugs Ltd, Middlesex House, 34-42 Cleveland Street, London W1P 6LB, United Kingdom. charlotte@cursci.co.uk
SO IDrugs, (1999) 2/9 (848-852).
ISSN: 1369-7056 CODEN: IDRUFN
CY United Kingdom
DT Journal; Conference Article
FS 037 Drug Literature Index
030 Pharmacology
LA English
SL English
AB Of the many sessions during the first day of the EPHAR meeting, several interesting topics emerged. Among these were a number of presentations investigating novel anti-inflammatory targets, including the search for a selective COX-2 inhibitor and the potential of cytokines/cytokine receptor targets (eg TNF.alpha.) as treatments for rheumatoid arthritis (RA) and other chronic inflammatory conditions. Recent advances in the understanding of the pathogenesis of diabetes and obesity have highlighted the need for a multi-therapeutic approach to treatment; several drugs in preclinical investigations were highlighted. Attention was drawn to the potential of AMPA/kainate receptors, historically investigated for the treatment of **neurodegenerative disease**, which are now showing promise as anti-ischemic therapeutics. Many novel therapeutics strategies were discussed in detail, including the CCK-B antagonists with considerable anxiolytic potential, mitochondrial mechanisms as targets for the treatment of **brain injury** and the use of stress-activated proteins in anti-ischemic research.

L10 ANSWER 10 OF 19 MEDLINE DUPLICATE 1
AN 2000103321 MEDLINE
DN 20103321
TI [Anti-TNF-alpha therapy as a new option in treatment of rheumatoid arthritis?].
Anti-TNF-alpha-Therapie **als** neue Option in der Behandlung der rheumatoiden Arthritis?.
AU Leeb B F; Sautner J
CS Niederosterreichischen Zentrum fur Rheumatologie am a. o. Krankenhaus Stockerau.. khstockerau@aon.at
SO WIENER MEDIZINISCHE WOCHENSCHRIFT, (1999) 149 (19-20) 554-7. Ref: 30
Journal code: XOU. ISSN: 0043-5341.
CY Austria
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 200007
EW 20000704
AB Due to intensive research in the field of cytokines during the last decade the knowledge of cytokine mediated processes has increased intensively. Modulation or even inhibition of the inflammatory cascade gave hope to effective therapeutic possibilities in sepsis or autoimmune diseases, particularly in rheumatoid arthritis (RA). Interestingly the application of biological immunomodulating substances could not increase the prognosis in sepsis, sometimes even deterioration occurred. However, in inflammatory bowel diseases and RA substantial efficacy could be revealed. Since blockade of II-1 or II-2 led to some beneficial results, but also sometimes to significant toxicity, TNF-alpha blockade gave hope to constitute a promising therapeutical target. Since the efficacy of a

monoclonal anti-TNF-alpha antibody and a recombinant soluble TNF receptor p75 fusion protein had been demonstrated in animal studies and in vitro, these results could be confirmed in controlled multicenter trials, showing significant improvement of patients according to Paulus and/or ACR criteria. However, a final assessment of therapeutical TNF-alpha blockade in RA cannot be given yet, since the tolerability in long-term application, particularly with respect to the risk of infections and the induction of malignancies and antibodies (e.g. drug induced lupus erythematosus) has to be observed carefully for longer times. Also the cost effectiveness of this new therapeutic approach needs further investigations.

L10 ANSWER 11 OF 19 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
AN 2000:45172 BIOSIS
DN PREV200000045172
TI Therapy with TNF-r Enbrel(R) results in remarkable symptom relief in patients (pts) with advanced primary amyloidosis (AL.
AU Juturi, Jaya (1); Karam, Mary A. (1); McLain, Denise A. (1); Murphy, Brian (1); Lutton, Suzanne (1); Hussein, Mohamad A. (1)
CS (1) Multiple Myeloma Program, Cleveland Clinic Cancer Center, Cleveland, OH USA
SO Blood, (Nov. 15, 1999) Vol. 94, No. 10 SUPPL. 1 PART 1, pp. 314a. Meeting Info.: Forty-first Annual Meeting of the American Society of Hematology New Orleans, Louisiana, USA December 3-7, 1999 The American Society of Hematology . ISSN: 0006-4971.
DT Conference
LA English

L10 ANSWER 12 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 2000078346 EMBASE
TI US drug and biologic approvals in 1998.
AU Spilker B.; FitzSimmons S.; Horan M.
CS Dr. S. FitzSimmons, 1100 15(th) Street NW, Washington, DC 20005, United States. sfitzsim@phrma.org
SO Drug Development Research, (1999) 48/4 (139-153). ISSN: 0272-4391 CODEN: DDREDK
CY United States
DT Journal; Article
FS 036 Health Policy, Economics and Management
037 Drug Literature Index
039 Pharmacy
LA English
SL English
AB The Prescription Drug User Fee Act of 1992 enhanced review resources for the Food and Drug Administration (FDA). The past 3 years have seen an unprecedented approval of 122 new drugs and 28 new biologics. Information is provided on the 39 new products approved by the FDA in 1998. (C) 1999 Wiley- Liss, Inc.

L10 ANSWER 13 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999088004 EMBASE
TI Disease modifying treatments for **multiple sclerosis**: What is on the horizon?.
AU Weilbach F.X.; Gold R.
CS Dr. F.X. Weilbach, Neurologische Universitatsklinik, Julius-Maximilians-Univ. Wurzburg, Josef-Schneider-Str. 11, D-97080 Wurzburg, Germany. f.weilbach@mail.uni-wuerzburg.de
SO CNS Drugs, (1999) 11/2 (133-157). Refs: 255 ISSN: 1172-7047 CODEN: CNDREF
CY New Zealand
DT Journal; General Review

FS 008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Stimulated by the successful introduction of interferon-.beta. as treatment for relapsing-remitting **multiple sclerosis** (**MS**) and based on an improved knowledge of the immunopathology of **MS**, a vast array of treatment options is currently under investigation for disease course modification. These are targeting relapse duration and intensity, relapse rate, disease progression and remyelination. The different approaches comprise mostly recombinant biotechnical agents, but also conventional immunosuppressants. Interferon-.beta. now can be regarded as an established disease modifying agent in relapsing remitting and secondary progressive **MS** as shown unequivocally in several well designed studies conducted by different pharmaceutical companies. Glatiramer acetate is also effective in relapsing remitting **MS**, although this conclusion is based on a lower level of evidence. A recent positive trial of mitoxantrone in chronic progressive **MS** underlines the efficacy of immunosuppression at least in subgroups of patients with **MS** who have high disease activity. Aside from the therapeutic approaches now already introduced into the clinical armamentarium, newer agents and treatment concepts include monoclonal antibodies, intravenous immunoglobulins, modulators of trimolecular complex and agents that interact with costimulatory molecules. Cytokine modulators and inhibitors of cell adhesion are promising candidates but their effect on the disturbed immunological network associated with **MS** has to be investigated thoroughly. In the future, simultaneous or sequential combinations of agents with different targets may significantly improve the efficacy of treatments for **MS**. The clinical evaluation of new treatment approaches will be difficult given the hetoregeneity and unpredictable course of the disorder. Interesting future therapeutic approaches include intracellular signal transduction modulators, vitamins and newer immunosuppressants. Gene therapy, vaccination with naked DNA or dendritic cells may also turn out to be useful. Besides developing new immunotherapies it seems indispensable to improve delivery of symptomatic treatment and rehabilitation aiming at the quality of life of individual **MS** patients. Identification of disease course predictors or treatment response will improve accuracy of therapeutic decision making.

L10 ANSWER 14 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999375527 EMBASE
TI Wyeth-Ayerst.
SO Formulary, (1999) 34/10 SUPPL. (103-107).
Refs: 8
ISSN: 1082-801X CODEN: FORMF
CY United States
DT Journal; General Review
FS 037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LA English

L10 ANSWER 15 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999375523 EMBASE
TI Schering-Plough.
SO Formulary, (1999) 34/10 SUPPL. (87-90).
Refs: 12
ISSN: 1082-801X CODEN: FORMF
CY United States
DT Journal; General Review
FS 037 Drug Literature Index

039 Pharmacy
LA English

L10 ANSWER 16 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999203664 EMBASE
TI A decade of biotech.
AU Stokes R.
CS . rstokes@compuserve.com
SO Pharmaceutical Technology Europe, (1999) 11/6 (66-67).
ISSN: 0164-6826 CODEN: PTEUFB
CY United Kingdom
DT Journal; Article
FS 027 Biophysics, Bioengineering and Medical Instrumentation
037 Drug Literature Index
LA English

L10 ANSWER 17 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999094417 EMBASE
TI New Drug Approvals of 1998 - Part 2.
AU Davis W.M.; Waters I.W.
CS Dr. W.M. Davis, Department of Pharmacology, Res. Inst. of Pharmaceut. Sciences, University of Mississippi, University, MS, United States
SO Drug Topics, (1999) 143/5 (58-74).
ISSN: 0012-6616 CODEN: DGTNA7
CY United States
DT Journal; General Review
FS 037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LA English

L10 ANSWER 18 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999393940 EMBASE
TI [Great future for small molecule].
GROSSE ZUKUNFT FUR KLEINE MOLEKULE.
AU Brunner U.
SO Pharmazeutische Zeitung, (4 Nov 1999) 144/44 (32-33).
ISSN: 0031-7136 CODEN: PZSED5
CY Germany
DT Journal; Note
FS 037 Drug Literature Index
039 Pharmacy
LA German

L10 ANSWER 19 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 2000031252 EMBASE
TI [The human body as effervescent source of new drugs].
KORPER **ALS** SPRUDELNDE QUELLE NEUER ARZNEISTOFFE.
AU Wolf E.
SO Pharmazeutische Zeitung, (23 Dec 1999) 144/51-52 (26).
ISSN: 0031-7136 CODEN: PZSED5
CY Germany
DT Journal; Note
FS 029 Clinical Biochemistry
037 Drug Literature Index
LA German